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Increases in Cerebrovascular Endothelial the Effects of Heritability Differences in Newborn Diseases

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Abstract

Vascular growth factor is a hallmark of cancer, and associations between genetic polymorphisms and neoplasia have been extensively studied in the adult population. Regarding the neonatal population, few studies have attempted to clarify the association of VEGF gene polymorphisms with neonatal pathologies, especially pathologies associated with late complications. Our aim was to review the literature on VEGF polymorphisms and neonatal morbidity. Narrative synthesis of results was performed using pre-determined subheadings (low birth weight or preterm infants, heart disease, lung disease, eye disease, brain disease, gastrointestinal disease). VEGF polymorphisms appear to be associated with neonatal pathologies. The involvement of VEGF and VEGF polymorphisms has been demonstrated in retinopathy of prematurity.

Keywords: Vascular endothelial growth factor (VEGF); Polymorphism; Neonatal diseases

Introduction

It is an important mediator of vascular permeability that is essential for fetal development. VEGF promotes the development of new blood vessels and subsequently increases the vascular permeability of endothelial cells [1].

Genetic polymorphisms of VEGF have been studied in adult populations, revealing its critical role in vascular growth and neoplasia development. It is highly polymorphic, exhibiting at least 30 functional single nucleotide polymorphisms (SNPs) in the 5'-untranslated region (UTR), 3'-UTR, and promoter regions. Genetic polymorphisms of VEGF have been studied not only in the adult population but also in the neonatal population [2].

Han Chinese adults reported that the VEGF gene 936 + C>T polymorphism may be an independent predictive marker for gastric cancer patients in a Korean population. Studies examining the association of VEGF polymorphisms with gastric and colon cancer have been inconclusive. A meta-analysis showed that the VEGF +936C>T gene polymorphism was significantly associated with an increased risk of developing gastrointestinal malignancies. Moderate importance of the VEGF-2578C/A polymorphism as a risk factor for bladder cancer was reported. A meta-analysis of 13 independent case-control studies involving a total of 4477 lung cancer patients found that the VEGF +936C/T, -460C/T, and -2578C/A polymorphisms were associated with lung cancer. No significant association was reported between 4,346 healthy controls [3].

The association between VEGF polymorphisms and diabetes in the adult population has attracted the attention of researchers. +936C/T (rs3025039) is likely associated with diabetic retinopathy susceptibility in Asian populations. It was shown that Gong and Sun showed an association between diabetic retinopathy and the -460T/C polymorphism of the VEGF gene, but not with the 2578C/A polymorphism. VEGF polymorphisms are associated with the development of macular edema in patients with diabetic eye disease and neovascular age-related macular degeneration [4].

Studies analyzing neonatal populations to assess associations between VEGF polymorphisms and pathology, especially late complications, are limited. Therefore, the aim of this study was to present a narrative synthesis of the research literature assessing the association between VEGF gene polymorphisms and neonatal morbidity [5].

Results

Pregnancy induced hypertension

Pregnancy is a complex physiological process involving numerous physiological and biochemical changes in the body to support fetal growth and development. Blood vessel development in the placenta initially implies angiogenesis and then extensive branching of fetal vessels through the interaction of angiogenesis and several regulatory factors. B. VEGF, placental growth factor (PGF), fibroblast growth factor (FGF), WNT, TGF-β, and members of the BMP family [6].

One of the key factors that play an important role in the development and maintenance of pregnancy is VEGF. This potent angiogenic factor stimulates the growth and proliferation of blood vessels, including the placental vasculature. This is essential for the proper supply of nutrients and oxygen to the developing fetus. Variations in VEGF expression and polymorphisms are associated with various pregnancy complications such as pregnancy-related hypertension (PIH), preeclampsia, and prematurity and low birth weight. Deletion of the VEGF gene results in severe defects in placental angiogenesis, leading to placental dysfunction and fetal death [7]. Mouse experiments have shown that ablation of the VEGF receptor is lethal to mouse fetuses, while overexpression of its soluble isoforms produces symptoms consistent with preeclampsia, such as hypertension, proteinuria, and intrauterine growth restriction (IUGR) shown to cause [8].

Pregnancy-related hypertension (PIH) is characterized by elevated blood pressure and can cause significant maternal and fetal morbidity and mortality. Defective angiogenic processes have been hypothesized to be responsible in part for microcirculatory vasoconstriction; the

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association between hypertension and angiogenesis, and the effect of genetic background on the incidence and severity of hypertensive disorders during pregnancy is suggested [9].

Pre-eclampsia is a serious complication that occurs after the 20th week of pregnancy, affecting approximately 2-8% of all pregnancies worldwide and is the leading cause of maternal death in developed countries. I'm here. The disease is characterized by hypertension, proteinuria, and organ damage, and can cause significant maternal and fetal morbidity and mortality.

Heart pathologies

Congenital heart disease, also known as congenital heart disease (CHD), is a group of structural heart defects that occur during fetal development. These defects are the most common type of birth defects, affecting approximately 0.8% to 1.2% of newborns worldwide [10].

The cause of CHD is complex and multifactorial, involving both genetic and environmental factors. A potential genetic factor implicated in CHD is mutation in the VEGF gene. VEGF plays an important role in cardiovascular development by promoting correct cardiac morphogenesis during the avascular phase of the heart and promoting blood vessel growth. The mechanisms by which VEGF polymorphisms contribute to the development of CAD are not fully understood. One possibility is that variations in VEGF expression and function could lead to abnormal growth and development of blood vessels in the developing heart, resulting in structural abnormalities that can lead to CAD. Another possibility is that VEGF polymorphisms may affect the fetal cardiac response to hypoxia, a common trigger of CAD. SNPs within the VEGF gene can affect VEGF expression and function and are associated with increased risk of several cardiovascular diseases, including CAD. In mouse embryos, VEGF overexpression and increased function were associated with TOF, pulmonary artery stenosis, and ventricular septal defect.

Digestive pathologies

Pathology of concern in the neonatal period is necrotizing enterocolitis (NEC). It is a complication with increased morbidity and mortality risk, especially in extremely preterm infants. The etiology is complex and involves multiple factors, including intestinal ischemia following fetal distress, intestinal hypo perfusion, infections that cause inflammatory processes in the mucosa, and infant formula intake. A role for genetics in the etiology of NEC is suggested by epidemiological data showing differences in susceptibility between population groups and twins. Genetic factors include VEGF polymorphisms. This result suggests that the G+405C VEGF polymorphism may be associated with increased risk of preterm. Suggests that there is Studies have observed decreased intestinal VEGF expression in human NEC and have shown that decreased VEGF signaling increases susceptibility to NEC in mouse models. On the other hand, in a cohort study (358 preterm infants), no association between VEGF C-2578-A and NEC was found.

Lung development and pathologies

Lung tissue has a high vascular content, which is important for gas exchange, so at significant levels he expresses VEGF. Therefore, optimal expression of VEGF is required for proper lung development as it contributes to the development of vasculature during embryonic development. VEGF functions as a mitogen and differentiation factor for various lung cells, including endothelial cells and type II lung cells. VEGF affects not only the vascular side but also the alveolar septal wall. This is because transient inactivation of her VEGF gene in the lung leads to cell apoptosis in the alveolar septal wall, enlarged voids and increased lung compliance. An important regulatory role of lung epithelial-expressed VEGF in function and development has been confirmed by animal models.

Conclusion

VEGF polymorphisms play important roles in placental and fetal development and determine neonatal pathology. They are associated with early-onset (cerebral hemorrhage, haemangioma, necrotic enteritis) and late-onset neonatal pathologies (retinopathy of prematurity, bronchopulmonary dysplasia). The involvement of VEGF and VEGF polymorphisms has been demonstrated in retinopathy of prematurity. However, further research is needed to better understand the mechanisms by which VEGF polymorphisms are involved in Functional genetic polymorphisms of angiogenic factors may affect the risk of developing bronchopulmonary dysplasia. Bronchopulmonary dysplasia is a disease with a multifactorial etiology in which premature arrest of lung growth is associated with other factors such as infection, oxygen-reactive species toxicity, and ventilator damage. In addition to these well-studied factors, it appears that decreased intestinal VEGF expression may increase susceptibility to necrotizing enterocolitis. Further studies examining this genetic susceptibility may improve treatment of neonates with known risk factors and have a positive impact on long-term outcomes. Gene expression and biochemical analyzes provide evidence for her VEGF causation in perinatal cerebral hemorrhage, a major complication that can lead to long-term disability. Advances in neonatal intensive care have improved survival for very preterm infants, who are most at risk for in vitro fertilization. Measuring her VEGF levels in these preterm infants can serve as an early predictor of the onset of cerebral hemorrhage and management of long-term sequelae. In conclusion, the association of VEGF polymorphisms with neonatal pathology is a worthy area to analyze in order to develop novel therapies with fewer side effects.

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